VOL. 35 NOVEMBER, 1907. NO. 11

THE
Journal
OF
Nervous and Mental Disease

Original Articles

A CASE OF APHASIA, BOTH "MOTOR" AND "SENSORY," WITH INTEGRITY OF THE LEFT THIRD FRONTAL CONVOLUTION: LESION IN THE LENTICULAR ZONE AND INFERIOR LONGITUDINAL FASCICULUS.*

BY F. X. DERCUM, M.D.,

PROFESSOR OF NERVOUS AND MENTAL DISEASE, JEFFERSON MEDICAL COLLEGE; NEUROLOGIST TO THE PHILADELPHIA HOSPITAL.

A new and all-absorbing interest has been given to the subject of aphasia by the views lately advanced by Pierre Marie. As revealed in his recent publications, Marie maintains that aphasia has been fundamentally misinterpreted and that this has resulted from purely theoretical considerations. Many writers indeed have taken as their point of departure complicated diagrams and schemes and have drawn from these long series of deductions. The results are just what might have been anticipated, for the prevailing view of aphasia is a doctrine essentially theoretical and schematic and which has been carried to such extremes as to find itself totally at variance with observed facts. To Marie the problem presented by aphasia is that of a disturbance of the intelligence, a disturbance which constitutes a special defect for the comprehension of language. Marie's views are absolutely opposed to the classical doctrine of aphasia. He denies that there exists in the left hemisphere an auditory verbal center in the foot or posterior half of the left first temporal convolution; he denies that there exists a visual verbal center in the left angular gyrus; he denies that there exists a center for writing in the foot of the left second frontal convolution; he denies that there exists in the foot of the left third frontal a motor verbal center. He insists that such an interpretation is merely schematic and theoretical and in no way justified by the facts. The theory of so-called word-deafness which is usually interpreted as a lesion in which auditory images are destroyed and in which the patient is no longer able to receive words in his auditory center and no longer capable of comparing new impressions with the auditory images already amassed and catalogued, he pronounces absolutely erroneous.

---

*Read at the meeting of the American Neurological Association, May 7, 8 and 9, 1907.

He points out that except in the rare cases in which non-comprehension of language is absolute, the patient can comprehend isolated words, short phrases, simple instructions, but in proportion as the phrases become lengthened, as the number of words becomes augmented and as the instructions become complicated, the comprehension of the patient fails. If, however, in testing such a patient we pronounce anew and separately each word, if we dissect the phrase into its elementary parts, if we decompose the instruction into its separate and distinct factors, we discover that the patient is again able to comprehend them. In short, that which disables or confuses the patient the most is the factor of complexity of language. Marie does not consider the zone of Wernicke as an auditory center—a so-called psycho-sensorial center—but as an intellectual center, and he insists upon the inevitable intellectual deficit which ensues when this center is destroyed. At the joint meeting of the Philadelphia and New York Neurological Societies, held in Philadelphia November 24th, 1906, in the discussion upon aphasia, I detailed the results of my studies of fourteen cases based upon the method of Marie. In all of these patients there was present an intellectual deficit. All revealed in a more or less pronounced degree an inability to carry out any but very simple instructions. Some failed absolutely, others complied, provided the instructions did not embrace more than two or possibly three factors. Only one or two could carry out instructions embracing four factors and always failed when this number was exceeded. How profound the intellectual disturbance is can readily be demonstrated when an aphasic is requested to do simple sums in arithmetic. Usually the additions are grossly incorrect and not infrequently the patient will begin his additions with the left-hand column, absolutely unconscious of the gross incongruity. Sometimes the intellectual deficit is glaringly revealed when the patient is instructed to perform some act with which he is long familiar and in the performance of which he has at one time been especially skillful. Thus Marie cites the case of one of his aphasics who, though sufficiently intelligent to mingle daily with his fellows and who, though formerly a chef de cuisine, proved to be utterly incapable of preparing so simple a dish as a fried egg. The patient having been taken to the kitchen and placed before the stove, was handed the necessary articles, a pan, an egg, butter, pepper and salt and was told what to do. The man hesitated for a moment and then commenced by breaking the egg very awkwardly. He emptied it into the pan without any precaution to avoid breaking the yolk, then he put some butter on top of the egg, sprinkled it with salt and pepper and then put the whole thing in the oven. Neither this gross blunder, nor the fact that the dish was absolutely unpresentable, disturbed the patient in the least.

As Marie clearly points out, the intellectual deficit in aphasia does not involve the intelligence as a whole. In general intellectual deficit may manifest itself in one of three ways: first, in the form observed in arrested cerebral development; secondly, in the form observed in dementia, as in paresis; and thirdly, in the special form seen in aphasia. Here the deficit instead of being generalized, involves merely one function or related group of functions. In other words, the deficit in aphasia is a special deficit dealing especially as Marie puts it, "with the stock of things acquired by didactic processes." Marie, it will be remembered, holds that aphasia is a unit, that it is not made up of sensory aphasia on the one hand or motor aphasia on the other, but that by lesion of the zone of Wernicke there is established an intellectual deficit for the comprehension of spoken language; that in so-called sensory aphasia or aphasia of Wernicke the lesion involves the zone of Wernicke (i. e., the supramarginal gyrus, the angular gyrus and the posterior portions of the two first temporal convolutions); that in so-called motor aphasia there is in addition to a lesion of this zone of Wernicke, also an involvement of the region of the lenticular nucleus. Lesion of the lenticular nucleus gives rise to anarthria, therefore in so-called motor aphasia, or Broca's aphasia, we have merely ordinary Wernicke aphasia plus anarthria.

How frequently aphasia is due to softening, the result of embolism or thrombosis, it is not necessary to point out, and Marie calls attention to the difference obtaining between cortical softening on the one hand and deep softening on the other. In cortical softening the extent of territory involved depends upon the point at which the middle cerebral artery has become obstructed. If the obstruction has occurred at or in advance of the point at which the branch or branches supplying the third frontal convolution are given off, the third frontal is of necessity included, but such involvement is by no means necessary to the production of aphasia. Not a small number of cases have been reported in which the third frontal alone has been involved in the softening without the slightest speech disturbance, as note the recent cases reported by Souques, and by Marie and Moutier. Again cases are not wanting in which the third frontal escaped involvement and in which notwithstanding motor aphasia was present. Such a case has only recently been placed on record by Marie and Moutier.

More interesting still are the facts in regard to the aphasia resulting from deep softening in which the cortex escapes and in which the lenticular zone and the white matter alone are involved. The case of aphasia that I here place on record belongs to this group. The case was as follows:

The patient was a man who was admitted to the nervous ward of the Philadelphia Hospital August 20th, 1903. His age was estimated at sixty-five. The diagnosis upon admission was that of aphasia with right hemiplegia. At the time it was learned that he had had an apoplectic seizure in the year 1902, after having previously suffered from attacks of dizziness, and that another attack of dizziness, followed by impairment of speech, had occurred in 1903. Owing to the fact that the patient could neither speak nor understand spoken language and that he could not understand writing, it was subsequently impossible to obtain either a family history and past medical history or a history of the present disease from him. It was noted that the right arm was more affected than the leg. He had great difficulty in articulation and enunciation. He had a very marked anarthria. When he attempted pantomime with the unparalyzed arm, his movements were confused.

Examined August 22nd, 1903, the diagnosis of right hemiplegia with aphasia was confirmed. The right upper limb was paralyzed and contractured, the forearm was flexed on the arm and the hand on the forearm. The right leg was paralyzed but not completely. The right foot and toes were contractured. There was also present a paresis of the lower half of the right side of the face. The patient could wrinkle his forehead well and equally on both sides, but he could not draw up the right angle of the mouth as well as the left. He closed both eyelids well, but did not close the right eye quite as well as the left. The

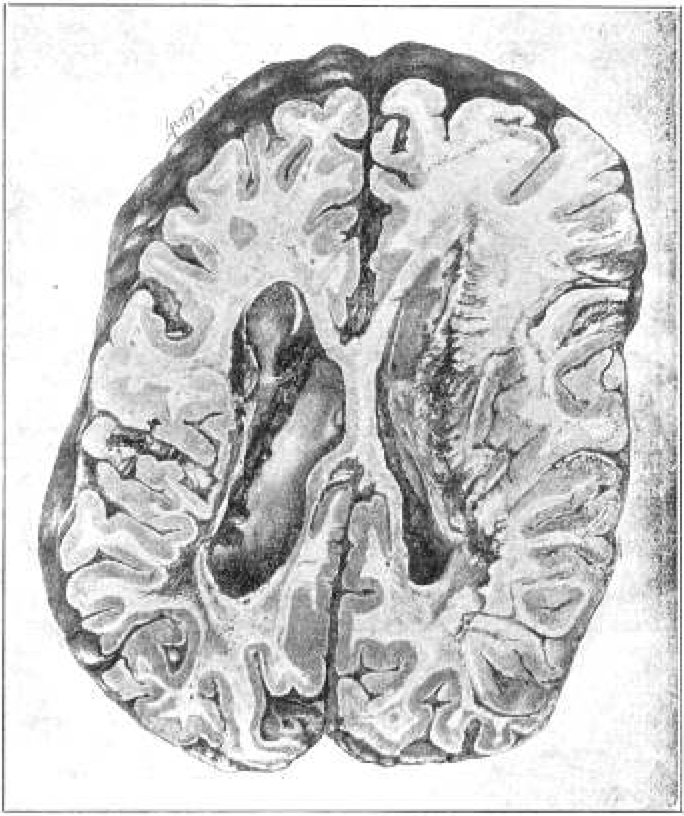

Fig. 1. Showing involvement of left lenticular nucleus and posterior longitudinal fasciculus; freedom of third frontal. Also extensive basal lesion on right side which immediately preceded death.

biceps, triceps and wrist reflexes were exaggerated upon the right side. They were about normal upon the left. The patellar reflex was exaggerated upon both sides. Ankle clonus was not elicited upon either side; this was also true of the tendo-Achillis jerk. The Babinski reflex was not positive upon either side. No sensory losses could be determined. The pupils were equal and contracted to light. Dr. Spiller, from whose record the above facts are abstracted, also noted that the patient had complete motor aphasia and that he was probably at least partially word deaf. He seemed to be able to give merely his name.

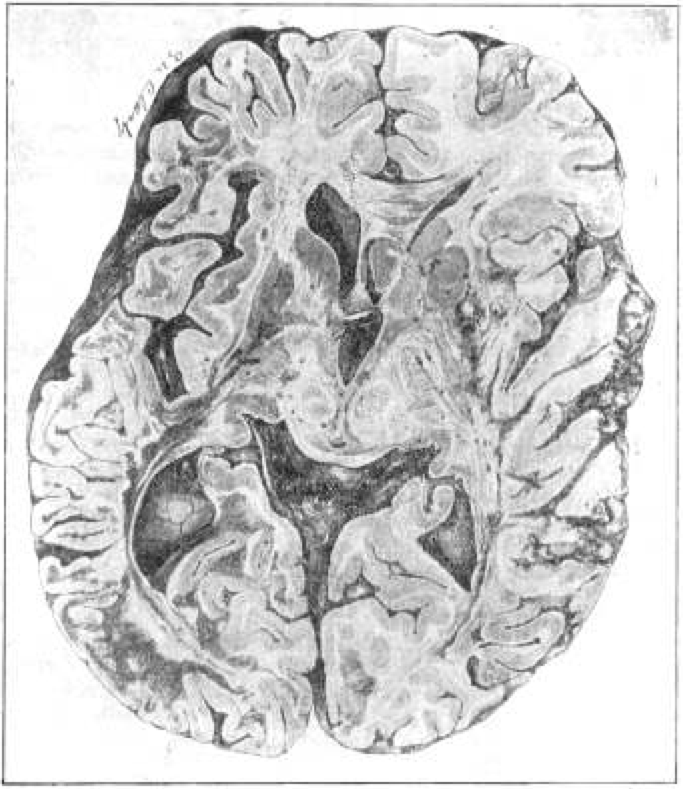

Fig. 2. ·Showing involvement of left lenticular nucleus and posterior longitudinal fasciculus; freedom of third frontal. Also extensive basal lesion on right side which immediately preceded death.

The patient remained in the wards enjoying comparative health and remained for some years in about the same condition. Early in November, 1906, he was repeatedly

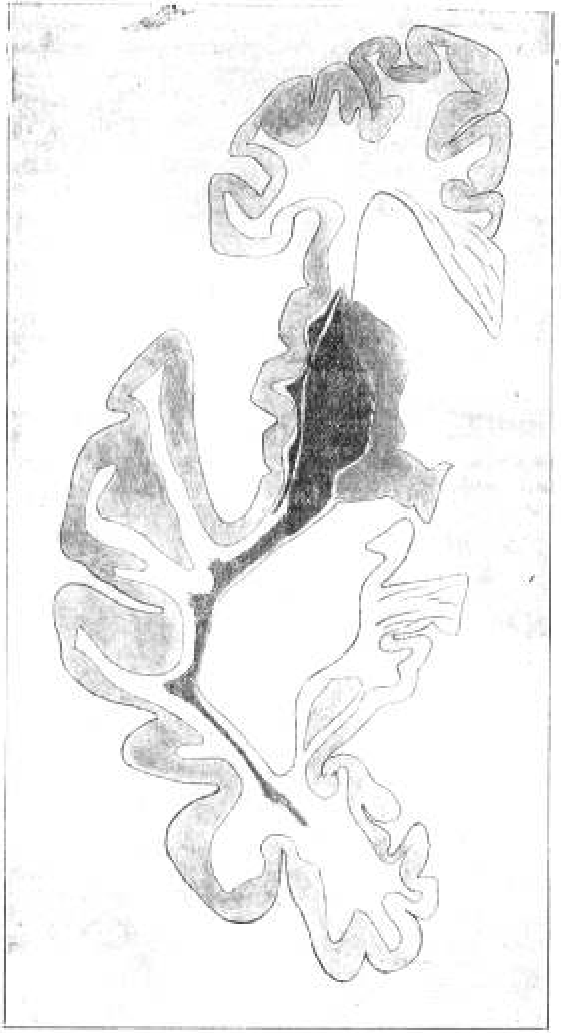

Fig. 3. Parallel Case of Marie.

Fig. 10. Horizontal section of the left hemisphere of Per . . . The softening met with here is of the type of deep softening * * * * It is represented by the black line which extends from R. to R', it occupies the external capsule and the base of the white substance of the convolutions of the island of Reil. At R' a line of softening bifurcates and is directed forward and inward toward the internal capsule; posteriorly the softening traverses the temporo-parietal isthmus and extends into the white substance of the temporo-occipital lobe R'' where it occupies the neighborhood of the external wall of the posterior horn of the lateral ventricle and follows faithfully its contour; it abandons this wall altogether posteriorly and terminates in the white substance of the occipital lobe. This patient was one of the most beautiful aphasics of Broca which it has been given me to observe. Here the aphasia of Broca has been produced because the lesion R R' has given rise to the anarthria and the lesion R' R'' to the aphasia. (Pierre Marie.)

examined by myself. He presented, of course, as before a right-sided hemiplegia with contractures, together with aphasia. It was clearly demonstrated that he was unable to understand what was said to him, that he was unable to comprehend or to carry out the simplest instruction unless this instruction was accompanied by gestures and pantomime on my part. Without such aid he failed utterly to comprehend what was desired of him. When told "raise your left hand," he failed utterly. If, however, the command were accompanied by my own gesture, he would at once comply. Under such instruction he would raise his hand, place it upon his head or other portions of his body, but in no instance could he perform such an act when instructed verbally. He was with great difficulty able to give his name, though the sounds were barely intelligible. He could also say "yes" and "no," though he substituted a "d" for the "n" in the "no." Upon one occasion during my tests he repeated after me the word "pencil," though very imperfectly. His anarthria was very pronounced. There was present also complete alexia; neither could he write or form letters with his left or unparalyzed hand; nor could he copy letters, though the tests were limited to his own name. There was also present apraxia. Pantomime was imperfectly preserved.

On December 5th, 1906, the patient was in his usual good health, but during the night suffered from a fresh apoplectic seizure, this time affecting the left side. The left arm and left leg became completely paralyzed and flaccid. There was conjugate deviation of the eyes and of the head to the right. The other and usual symptoms of an apoplectic seizure were present; the patient was comatose, presented stertorous breathing and a rapid pulse of rather high tension. There was little change in his condition subsequently. The deviation of the head and eyes to the right became somewhat less pronounced and he became somewhat weaker. Congestion and edema of the lungs gradually made their appearance and the patient died on December 10th at 10 p. m.

Autopsy.—The results of the general pathological examination need not detain us here. They consisted in brief of adherent pericardium, hypertrophy and dilatation of the heart, fibroid myocarditis, congestion of the lungs and interstitial nephritis. Examination of the brain proved to be of great interest. There was no gross lesion of any convolution. A horizontal section passing through the basal ganglia at the level of the foot of the left third frontal convolution revealed two extensive lesions. First an old lesion upon the left side of the brain involving the lenticular zone and the inferior longitudinal fasciculus; secondly, a recent and very extensive softening upon the right side involving the basal ganglia and capsule. The lesion upon the right side was of course the one which immediately preceded death; it is the old lesion upon the left side which concerns us here. As just stated, the lesion upon the left side involves the lenticular nucleus, the adjacent portion of the internal capsule and can also be traced far back along the inferior longitudinal fasciculus. The third frontal convolution is absolutely intact, as is also its subjacent white matter. This is also true of the convolutions of the regions of Wernicke and of the angular gyrus. This interesting case is almost an exact parallel of the case described by Marie in his article in the *Semaine Médicale,* May 23rd, 1906, and illustrated by Figure 10. This figure is here reproduced. Marie says of his case that the patient presented one of the most beautiful aphasias of Broca—motor aphasia—which it had ever been given him to observe.

The question arises, how can such a lesion as is here described give rise to aphasia—to motor aphasia or the anarthria of Marie upon the one hand and to sensory aphasia or aphasia of Wernicke on the other? The lesion of the lenticular zone readily explains according to Marie's view the anarthria. It would seem further that the degeneration of the posterior longitudinal fasciculus produces sensory or Wernicke aphasia by isolating the zone of Wernicke and secondly the degeneration of this fasciculus also produces alexia because it cuts off all communication between the zone of Wernicke and the visual centers. To me this case has been one of great interest for it is most suggestive. We know very little of the function of the striated body. It is a structure which is persistent throughout all the vertebrate forms and in the lower vertebrates, as is well known, it constitutes all of the cerebrum. Even when we ascend the scale as far as birds we find that in them the pallium,—the part corresponding to our cortex,—is still very rudimentary and that the striated bodies must carry on the cerebral function. In man the striated body has retained so great a size that it cannot possibly be regarded as a rudimentary or vestigial organ. In spite of the enormous development of the pallium, this organ has persisted in the higher vertebrates to such a degree as to necessitate the inference that it has most important functions. That the striated body should be concerned in speech presents nothing inherently improbable. While I do not mean to compare the speech of the parrot with the speech of man, the facts justify the inference that the parrot talks with his striated body; this view has also been expressed by Kalischer. Unfortunately physiologists have thus far given us but little information as to the function of the striated body; it is possible that the propulsive movements of Majendie, the circus movements of Nothnagel have something to do with movements of co-ordination. However, the facts of the special disturbances claimed by Majendie and by Nothnagel have been questioned and denied and we are of necessity forced to draw our inferences from pathological and clinical evidence.

The function performed by the lenticular nucleus appears to be one of co-ordination of complex muscular movements, and motor speech is pre-eminently a function requiring the co-ordination of complex movements; e. g., the movements of the tongue, palate, lips, larynx and of the muscles concerned in expiration. A derangement of this intricate co-ordination means of necessity anarthria, means of necessity the impossibility of speech enunciation. Just in proportion as the substance of the lenticula is destroyed, so must there be an absence of motor speech; just in proportion as the function of the lenticula is deranged, so must there be present an anarthria.

REFERENCES.

Pierre, Marie. La Semaine Médicale, May 23, 1906. Ibidem, Oct. 17, 1906.

Pierre Marie. La Semaine Médicale, Nov. 28, 1906.

Pierre Marie et François Moutier. Bulletins et Mémoires de la Société Médicale des Hôpitaux de Paris, Decembre 14th, 1906.

Pierre Marie et François Moutier. Bulletins et Mémoires de la Société Médicale des Hôpitaux de Paris, Fevrier, 15th, 1907.

Pierre Marie. La Revue de Philosophie, 1907.

Pierre Marie. La Presse Médicale, No. 4, 12 Janvier, 1907.

Souques. La Presse Médicale, October 24th, 1906, p. 683.

Souques. Bulletin et Mémoires de la Société Médicale des Hôpitaux de Paris, 19 Octobre, 1906.

Souques. Bulletins et Mémoires de la Société Médicale des Hôpitaux de Paris, 1 Mars, 1907.